

Soil Ingestion: A Concern for Acute Toxicity in Children

Edward J. Calabrese,¹ Edward J. Stanek,¹ Robert C. James,² and Stephen M. Roberts²

¹School of Public Health, University of Massachusetts, Amherst, MA 01003 USA; ²Center for Environmental & Human Toxicology, University of Florida, Gainesville, FL 32611 USA

Several soil ingestion studies have indicated that some children ingest substantial amounts of soil on given days. Although the EPA has assumed that 95% of children ingest 200 mg soil/day or less for exposure assessment purposes, some children have been observed to ingest up to 25–60 g soil during a single day. In light of the potential for children to ingest such large amounts of soil, an assessment was made of the possibility for soil pica episodes to result in acute intoxication from contaminant concentrations the EPA regards as representing conservative screening values (i.e., EPA soil screening levels and EPA Region III risk-based concentrations for residential soils). For a set of 13 chemicals included in the analysis, contaminant doses resulting from a one-time soil pica episode (5–50 g of soil ingested) were compared with acute dosages shown to produce toxicity in humans in clinical studies or case reports. For four of these chemicals, a soil pica episode was found to result in a contaminant dose approximating or exceeding the acute human lethal dose. For five of the remaining chemicals, the contaminant dose from a soil pica episode was well within the reported dose range in humans for toxicity other than lethality. Because both the exposure episodes and the toxicological response information are derived from observations in humans, these findings are regarded as particularly relevant for human health risk assessment. They suggest that, for some chemicals, ostensibly conservative soil criteria based on chronic exposure using current EPA methodology may not be protective of children during acute soil pica episodes. *Key words:* acute toxicity, exposure assessment, pica, risk assessment, soil ingestion.

Environ Health Perspect 105:1354–1358 (1997). <http://ehp.niehs.nih.gov>

When evaluating risks posed by contaminated soil, incidental soil ingestion is often the most important pathway of exposure. For purposes of estimating risks to children, the EPA [J.W. Porter, unpublished data; (1)] assumes that most children ingest relatively small quantities of soil (e.g., <100 mg/day), while the upper 95th percentile are estimated to ingest 200 mg/day on average. This latter figure has been frequently employed as the assumed soil ingestion rate for children, both in estimating risks from soil contaminants under a residential land use scenario and in setting risk-based cleanup goals. While risk assessments for contaminated sites are directed principally to public health concerns for long-term exposure, the EPA has conceptually addressed the possibility that some children may display, at least on occasion, profound soil ingestion (referred to as soil pica) in quantities far greater than the upper 95th percentile value. For such children, the EPA (2) has proposed that risk assessors assume soil ingestion at a rate of 5 g soil/day. This is routinely ignored in practice, however, and risks from soil pica are rarely addressed explicitly in risk assessments.

Recently, there has been considerable effort by the EPA and state environmental regulatory agencies to define acceptable risk-based levels of contaminants in soils. The New Jersey Department of Environmental Protection was one of the first regulatory agencies to attempt to promulgate comprehensive risk-based soil standards, and in

1992 this department proposed standards for about 100 contaminants in soils (3). The methods used to derive the proposed soil standards were generally consistent with contemporary risk assessment practice, and the values were intended to be health protective for individuals, including children, under circumstances in which the property is used for residential purposes. The proposed soil standards were derived based on the potential for chronic exposure and, consistent with EPA recommendations, a soil ingestion rate of 200 mg soil/day for children was employed. An analysis was subsequently conducted to determine whether the proposed standards would also be health protective under circumstances of shorter, more extensive soil exposure, as might occur with soil pica. The analysis concluded that adverse human health effects were possible from acute or subchronic ingestion of 5 g soil at the proposed standard for nearly 42% of the chemicals and that there was the potential for toxicity from ingestion of as little as 200 mg soil for 17 of these chemicals (4).

For a variety of reasons, the soil standards proposed by the New Jersey Department of Environmental Protection were never implemented. Several states have, however, developed similar lists of soil contaminant concentrations to use as screening tools for sites, and the EPA has recently released its *Soil Screening Guidance*

(5), which provides residential land use risk-based soil concentrations for about 100 chemicals. In general, these soil guidance concentrations are intended to be broadly applicable and conservative and represent safe levels of the contaminants in soils, even under circumstances in which children may have extensive soil contact, such as a back yard, playground, or day care facility.

Our objective for this study was to make a preliminary assessment of the risks posed by soil contaminants at contemporary guidance concentrations when there is soil pica. While addressing the same basic issue—the health protectiveness toward children of soil standards or guidance concentrations—the analysis differed from that conducted previously by Technical Resources, Inc. (TRI) (4) in several important respects. First, we based estimates of soil ingestion during a soil pica episode on observations from other soil ingestion studies (6–10). As discussed below, these studies indicate that soil pica episodes may involve soil quantities much greater than 5 g. Second, the basis for comparison is different: while there are sets of soil criteria available from various states, we selected the EPA *Soil Screening Guidance* concentrations (5) so that the analysis might have relevance from a national perspective. For chemicals without a soil criteria value listed in this source, we used the EPA Region III risk-based soil concentrations for residential land use (11). Finally, the emphasis on the source of toxicity information was somewhat different from that employed previously; many of the conclusions regarding acute and subacute risk in the TRI analysis were based on toxicity values extrapolated from animal data, with the inclusion of substantial uncertainty factors. To avoid the uncertainty inherent in extrapolation of animal data to humans, we used only acute toxicity information derived from clinical studies or case reports in this analysis.

Magnitude and Variability of Soil Pica

Realistic estimates of soil pica are problematic. Estimating the frequency, magnitude, variability, and duration of soil pica has not

Address correspondence to E.J. Calabrese, School of Public Health, University of Massachusetts, Morrill, N344, Amherst, MA 01003 USA.

Received 2 July 1997; accepted 4 September 1997.

been the object of extensive research. In the course of three soil ingestion studies, we have observed unambiguous soil pica in two children. One child was observed to ingest 20–25 g soil on 2 of 8 days (7,12). A second child displayed more consistent but less striking soil pica in which high soil ingestion (~1–3 g/day) was observed on 4 of 7 days (8). A 1988 study by Wong (9) noted soil pica (>1.0 g/day) in 5 of 24 children of normal mental capability on at least 1 of 4 days (i.e., 1 day of observation per month for 4 months). Nine individual subject-day values out of 84 (10.5%) had soil ingestion estimates >1 g/day. One mentally retarded child displayed consistent massive soil ingestion over the 4 days of 48.3, 60.7, 51.4, and 3.8 g soil. These data suggest that soil pica may vary considerably both between and within individuals and are consistent with observations that generalized pica behavior is common in normal children, but may be more prevalent and of longer duration in mentally retarded children (9).

Soil ingestion studies had very limited durations, usually for about a week or less. Consequently, it has not been possible to obtain a clear understanding of intraindividual variability in soil ingestion activity. Nonetheless, several years after the publication of our initial soil ingestion study in children (6), we developed a methodology to estimate daily soil ingestion in study children (13). This allowed the estimation of up to eight different daily measures of soil ingestion (i.e., a separate estimate for each day of the study) per subject in the original study. Using the median soil ingested for each study child and the standard deviation of these estimates (assuming a log-normal distribution for soil ingestion), we simulated soil ingestion for 365 days for each child and tabulated the frequency of soil pica days (>1 g/day) (13). This model-based prediction indicated that the majority (62%) of children will ingest >1 g soil on 1–2 days/year, while 42% and 33% of children were estimated to ingest >5 and >10 g soil on 1–2 days/year, respectively. These model-based estimates were qualitatively significant because they suggest that soil pica is not restricted to a very small percentage of the normal population of children, but may be expected to occur in a sizable proportion of children throughout the course of the year. The findings also support the hypothesis that there is considerable interindividual variation with respect to soil pica frequency and magnitude. Thus, for the majority of children, soil pica may occur only on a few days of the year, but much more frequently for others. If soil pica is seen as an expected,

although highly variable, activity in a normal population of young children, rather than an unusual activity in a small subset of the population, its implications for risk assessment become more significant.

Relating Soil Pica to Hazard Potential

Thirteen chemicals were selected for the analysis based on the availability of acute human toxicity data and on the suggestion in the TRI study (4) that acute toxicity problems may exist for those chemicals. These chemicals were antimony, arsenic, barium, cadmium, copper, cyanide, fluoride, lead, naphthalene, nickel, pentachlorophenol, phenol, and vanadium. For each of these chemicals, information was sought regarding acute dosages producing lethality, as well as the lowest dosage reported to produce significant nonlethal effects. For the most part, these dosages came from case reports of intoxication following accidental ingestion of the chemical in question. Cases involving ingestion of more than one substance were not considered, given the obvious potential for confounding of the dose–toxicity relationship for the chemical in question. Doses reported to produce acute toxicity were compared with those that would result from acute ingestion by a small child of 5, 25, or 50 g soil containing the chemical at the EPA screening concentration (Table 1) (14–32). To facilitate comparisons, all doses are expressed in terms of milligram per kilogram body weight. Toxic dosages from case reports, in some instances, had to be derived using an assumed body weight based on the description of the subject(s). For the pica child, a 13-kg body weight is assumed, which closely corresponds to the 50th percentile body weight of a 3-year-old child (33).

As shown in Table 1, in the case of arsenic, a pica episode involving soil contaminated at the screening level value would result in an ingested dose of 2, 8, or 15 µg/kg, depending upon whether the child ingests 5, 25, or 50 mg of soil, respectively. The highest of these dosages is well below acute doses identified in our literature survey as associated with toxicity. Similarly, projected doses of antimony, naphthalene, and pentachlorophenol from a soil pica episode involving soil at the screening level were also less than those reported to produce acute toxicity. For the remaining chemicals, however, the amount contained in 5–50 g of soil is within the reported toxic range in humans. In fact, for cyanide, fluoride, phenol, and vanadium, the ingested dose from 25 g of soil exceeds amounts reported to produce lethality.

Discussion

Risk-based soil screening levels and clean-up goals are currently developed based on chronic exposure. The implicit assumption is that contaminant concentration limits that are health protective under chronic exposure circumstances will be protective also for acute exposure. While there is a certain logic to this assumption, it may not be valid when the acute exposure is much larger than the time-averaged chronic exposure. Soil ingestion rates in children appear to provide an excellent example of this situation. While 95% of small children may ingest, on average over time, 200 mg soil/day or less, their soil ingestion behavior can include episodic ingestion of 250 times that amount or more. In establishing soil screening levels and clean-up goals for exposure scenarios that can include contact with soils by small children, it seems reasonable to take this behavior into consideration.

The relatively simple analysis presented here is intended to be preliminary, focusing on a limited group of chemicals, and probably does not address all of the acute toxicity endpoints that may be of potential concern. The results strongly suggest that current methodology for calculating risk-based soil screening levels and clean-up goals based on chronic exposure assumptions may not adequately protect children exhibiting soil pica behavior from acute toxicity from some chemicals. Depending upon the magnitude of soil ingested and the specific contaminant, a soil pica episode may result in the ingestion of doses similar to, or greater than, those observed in clinical reports to produce severe toxicity, including death. While comparisons in this study were based on EPA-derived soil screening values, it should be noted that many states have also developed lists of risk-based soil concentrations using methodology that is similar, for the most part, to that used by the EPA. It is logical to suspect that concerns about the health protectiveness of current soil criteria are relevant to these values as well.

It is important to acknowledge the caveats associated with this analysis. Dose–response data for acute toxicity in humans are generally quite limited, particularly for children. By and large, acute toxicity data come principally from case reports of accidental ingestion in which dose estimation may be uncertain. In situations where a range of doses associated with toxicity has been reported in the literature, the lowest doses were used in the analysis to provide an indication of the dose required for toxicity. In situations where data are extremely limited (e.g., only a few case reports exist), even

Table 1. Estimates of acute toxicity associated with soil pica episodes in young children at EPA soil screening concentrations

Chemical	Soil screening ^a value (mg/kg soil)	Soil intake (g soil/event)	Dose from soil ^b (mg/kg body weight)	Lethal dose (mg/kg body weight)	Reference	Nonlethal toxic dose (mg/kg body weight)	Effects	Reference
Antimony	31	5 25 50	0.01 0.06 0.12	ND	—	0.528	Nausea, vomiting	(14)
Arsenic	0.4 ^c	5 25 50	0.002 0.008 0.015	1–3	(16)	1	Throat irritation, nausea and vomiting	(15)
Barium	5,500	5 25 50	2.1 10.6 21.2	43–57	(17)	2.86–7.14	Acute threshold for toxicity in adults	(16)
Cadmium	78	5 25 50	0.03 0.15 0.30	25	(18)	0.043–0.07	GI irritation and vomiting in children	(18,19)
Copper	3,100*	5 25 50	1.2 6.0 11.9	14–429	(21)	0.09	Vomiting and diarrhea	(21)
Cyanide	1,600	5 25 50	0.6 3.1 6.2	0.5	(23)	ND		—
Fluoride	4,700*	5 25 50	1.8 9.0 18.1	4	(24)	0.04–3.0 ^d	GI effects	(24)
Lead	400	5 25 50	0.2 0.8 1.5	ND	—	0.02	Decreased ALAD	(25)
Naphthalene	3,100	5 25 50	1.2 6.0 11.9	ND	—	~70 ^e 109	Severe bladder pain and near blindness Hemolytic anemia	(26,27)
Nickel	1,600	5 25 50	0.6 3.1 6.2	570	(29)	0.009 ^e	Contact dermatitis	(29)
PCP	3	5 25 50	0.001 0.006 0.012	17 ^f	(31)	ND		—
Phenol	47,000	5 25 50	18.1 90.4 180.8	39 ^f 10–50 ^g	(31,32)	14	GI effects	(31)
Vanadium	550	5 25 50	0.2 1.1 2.1	0.86	(33)	ND		—

Abbreviations: ND, not determined (no acute toxicity doses in humans were identified); GI, gastrointestinal; ALAD, aminolevulinic acid dehydratase; PCP, pentachlorophenol.

^aValues with an asterisk are from the EPA's *Risk-based Concentration Tables, Region III* (11); values without an asterisk are from the EPA's *Soil Screening Guidance* (5).

^bCalculated as (soil screening value × soil intake)/13 kg assumed body weight.

^cThis value may be below background levels in some parts of the United States. In such cases, the natural background value would be used.

^dEstimated dose based on an assumed body weight of 35 kg.

^eEstimated dose based on an assumed body weight of 70 kg.

^fEstimated dose based on an assumed body weight of 59 kg.

^gEstimated dose based on an assumed body weight of 5 kg for an infant.

the lowest value of the reported range may overestimate the dose needed to produce toxicity. This is because individual cases do not measure the dose needed to produce a toxic effect such as death; they only indicate that the necessary dose was exceeded, and the lowest among the case reports may be well in excess of the threshold for the toxic effect of concern. On the other hand, the lowest value may reflect a response by an

unusually sensitive individual or special circumstances not generally applicable. Information in the literature regarding toxic but survived doses or no-effect doses in sizable populations of individuals would be helpful in gaining perspective on toxic doses, but are seldom available for acute exposure among humans to environmental chemicals.

Only one of the comparisons was based on toxicity data from individuals known to

be sensitive to the toxicant—contact dermatitis from ingestion of nickel in nickel-sensitized subjects. Among these individuals, dermal reactions can occur following ingestion of very small amounts of nickel (34). For some of the other toxicants, the toxicity value used for comparison may not encompass all of those with special sensitivity. For example, in the case of naphthalene, the estimated exposure from ingestion of as much as

50 g contaminated soil was still well below the reported, frankly toxic oral human dose. However, it is generally accepted that there is considerable interindividual variation in susceptibility to naphthalene-induced hemolysis. Individuals with a glucose-6-phosphate dehydrogenase (G-6-PD) deficiency, a red blood cell condition found in 13% of American black males, are known to have enhanced susceptibility to naphthalene (35–37). In addition, infants are considered very sensitive to the hemolytic effects of naphthalene, possibly due to their reduced capacity to conjugate and excrete the chemical (37). In the case of copper, the acute dose used in the comparison table for nausea, vomiting, and diarrhea is from poisonings in adults. There is evidence from a number of case reports that infants and children under 10 years of age are particularly susceptible to gastrointestinal effects from copper in drinking water (38), and this increased sensitivity may be applicable to acute ingestion of copper as well.

The dose estimates used in this analysis are ingested doses rather than absorbed doses, and it is possible that matrix effects of contaminants in soils may retard absorption and thereby mitigate their toxicity to some degree. The extent to which this may occur is difficult to evaluate because reliable data on bioavailability from soils are available for very few chemicals (39). From a toxicological perspective, the expectation that absorption from soils may be diminished is counterbalanced in a number of instances by the severity of the toxic endpoint. For example, even if matrix effects reduced the absorbed dose of chemicals such as cyanide, fluoride, phenol, and vanadium to below lethal levels, serious toxicity could nonetheless result.

The frequency with which children experience acute poisoning from ingestion of contaminated soils is unknown. Quinby and Clappison (40) described a case in which a child became severely intoxicated following ingestion of parathion in contaminated soil, but such reports are rare in the literature. Conceivably, this could reflect, in part, a failure of parents and medical personnel to associate acute illness with soil pica except in obvious cases. Similarly, the likelihood of acute intoxication from consumption of contaminated soil is difficult to predict and is, of course, dependent on the occurrence of a soil pica event at a location with significantly contaminated soil. For example, in the case of the soil pica child who was observed to ingest 20–25 g soil on two occasions (7,12), the levels of lead in her yard were 20–25 ppm. However, if she had ingested soil that had 500–1,000 ppm lead, which is common in some older inner cities, the biological impact may have been more profound,

resulting in a substantial increase in the blood lead level according to the EPA biokinetic uptake model for lead (10). Thus, the possibility of intoxication is complex, being affected by the frequency and magnitude of the pica event, access to contaminated soil, and also the quality of adult supervision.

In addition to interindividual differences in susceptibility to toxic substances, there are likely to be important differences in soil pica activities as well. Within this context, young children have little awareness of the concept of contamination or disgust concerning things they ingest; they also have incomplete knowledge of edible and inedible substances (41–44). Soil ingestion and other pica activity in young children then may not reflect aberrant behavior as much as behavior that declines as care giver socialization efforts and children's sensory discriminations and cognitive advances coalesce to dampen its exercise. Such an explanation also would help to account for the frequent observation that pica activity occurs among the mentally retarded (45–48). These observations reinforce the massive and consistent episodes of soil pica in a mentally retarded child as reported by Wong (9).

The analysis presented here is based exclusively on observations in humans, both in terms of soil pica behavior and doses associated with toxicity. While there are acknowledged limitations in the analysis, as discussed above, two of the greatest sources of uncertainty common to most toxicological evaluations are absent, that is, extrapolation of data from animals to humans and extrapolation of dose beyond the observed range. The selective use of human data contributes to greater confidence in the relevance of the analysis to human health and, at the same time, greater concern for its implications. Given the serious nature of acute toxicity potentially associated with consumption of contaminated soils during a soil pica episode, this analysis suggests that greater attention must be paid by regulatory and public health agencies to this issue when developing health-based criteria and standards for soils. There should also be more careful and explicit consideration of this possibility in risk assessments where contaminated soil and the potential for present or future exposure by children exist.

REFERENCES

1. U.S. EPA. Exposure Factors Handbook. Washington, DC:U.S. Environmental Protection Agency, 1996.
2. U.S. EPA. Superfund Health Assessment Manual. Washington DC:U.S. Environmental Protection Agency, 1985.
3. Site Remediation Program, cleanup standards for contaminated sites, proposed new rules: N.J.A.C. (Trenton) New Jersey Register, 3 February 1992;373–403.
4. Technical Resources, Inc. Evaluation of acute and subchronic toxicity information for environmental contaminants. Rockville, MD: Technical Resources, Inc., 1994.
5. U.S. EPA. Soil Screening Guidance: Technical Background Document. EPA/540/R-95/128. Washington DC:U.S. Environmental Protection Agency, 1996.
6. Calabrese E, Barnes R, Stanek EJ III, Pastides H, Gilbert CE, Veneman P, Wang X, Lasztity A, Kostetki PT. How much soil do young children ingest: an epidemiological study. *Regul Toxicol Pharmacol* 10:123–137 (1989).
7. Calabrese EJ, Stanek EJ, Gilbert CE. Lead exposure in a soil pica child. *J Environ Sci Health A28*:353–362 (1993).
8. Calabrese EJ, Stanek EJ, Barnes R. Soil ingestion rates in children identified by parental observation as likely high soil ingesters. *J Soil Contam* 6(3):271–279 (1997).
9. Wong MS. The role of environmental and host behavioral factors in determining exposure to infection with *Ascaris lumbricoides* and *Trichuris trichiura*. [Ph.D. thesis]. University of the West Indies, Mona, Jamaica, 1988.
10. Calabrese EJ, Stanek E. Soil pica: not a rare event. *J Environ Sci Health A28*:373–384 (1993).
11. U.S. EPA. Risk-Based Concentration Tables, Region III. Philadelphia, PA:U.S. Environmental Protection Agency, 1996.
12. Calabrese EJ, Stanek E, Gilbert CE. Evidence of soil-pica behavior and quantification of soil ingestion. *Hum Exp Toxicol* 10:245–249 (1991).
13. Stanek EJ III, Calabrese EJ. Daily estimates of soil ingestion in children. *Environ Health Perspect* 103:276–285 (1995).
14. Dunn JT. A curious case of antimony poisoning. *Analyst* 531:532–533 (1928).
15. Armstrong CW, Stroube RB, Rubio T, Siudyla EA, Miller GB. Outbreak of fatal arsenic poisoning caused by contaminated drinking water. *Arch Environ Health* 39:276–290 (1984).
16. Reeves AL. Barium. In: *Handbook on the Toxicology of Metals* (Friberg L, Norberg GF, Vouk VB, eds). 2nd ed. New York:Elsevier Science Publishers, 1986;84–94.
17. Wisniewska-Knypl JM, Jablonska J, Myslak Z. Binding of cadmium on metallothionein in man: an analysis of fatal poisoning by cadmium iodide. *Arch Toxicol* 28:46–55 (1971).
18. Lauwerys R. Cadmium in man. In: *The Chemistry, Biochemistry and Biology of Cadmium* (Webb M, ed). New York: Elsevier/North Holland Biomedical Press, 1979;433–453.
19. Nordberg G, Slorach S, Stenstrom T. Kadmiumförgiftning orsakad av kalldrycksautomat (Cadmium poisoning caused by a cooled-soft-drink machine.) *Lakartidningen* 70(7):601–604 (1973).
20. Chuttani HK, Gupta PS, Bulati S, Gupta DN. Acute copper sulfate poisoning. *Am J Med* 39:849–854 (1965).
21. Wyllie J. Copper poisoning at a cocktail party. *Am J Public Health* 47:617 (1957).
22. Gettler AO, Blaine JO. The toxicology of cyanide. *Am J Med Sci* 195:182–198 (1938).
23. Dukes NGH. Fluoride. In: *Side Effects of Drugs. Annual 4*. Oxford:Excerpta Medica, 1980;354.

24. Hoffman R, Mann J, Calderone J, Trumbull J, Burkhardt M. Acute fluoride poisoning in a New Mexico elementary school. *Pediatrics* 65: 897-900 (1980).
25. Stuk EJ. Biological response of male and female volunteers to inorganic lead. *Int Arch Arbeitsmed* 33(2):83-97 (1974).
26. Grant WM. *Toxicology of the Eye*. Springfield, IL:Thomas, 1986.
27. Gidron E, Leurer J. Naphthalene poisoning. *Lancet* 1:228-230 (1956).
28. Daldrup T, Haarhoff K, Szathmary SC. Toedliche nickelsulfateintoxikation (Fatal nickel sulfate poisoning). *Beitrage zur Gerichtlichen Medizin* 41:141-144 (1983).
29. Dreisbach RH. *Handbook of Poisoning: Prevention, Diagnosis, and Treatment*. 10th ed. Los Altos, CA:Lange Medical Publications, 1980.
30. Deichman WB. *Toxicology of Drugs and Chemicals*. New York:Academic Press, Inc. 1969.
31. Stokinger HE. The Metals. In: *Industrial Hygiene and Toxicology Vol IIA* (Patty FA, ed). 3rd ed. New York:John Wiley and Sons, 1981;2013-2032.
32. Calabrese EJ, Stanek EJ. High levels of exposure to vanadium by children aged 1-4. *J Environ Sci Health*, A28(10):2359-2371 (1993).
33. U.S. EPA. *Exposure Factors Handbook*. Washington DC:U.S. Environmental Protection Agency, 1989.
34. Cronin E, Dimichiel AD, Brown SS. Oral challenge in nickel-sensitive women with hand eczema. In: *Nickel Toxicology* (Brown SS, Sunderman FW Jr, eds). New York:Academic Press, 1980;149-152.
35. Dawson JP, Thayer WW, Desforges JF. Acute hemolytic anemia in the newborn infant due to naphthalene poisoning: report of two cases, with investigations into the mechanism of the disease. *Blood* 13:1113-1125 (1958).
36. Shannon K, Buchanan GR. Severe hemolytic anemia in black children with glucose-6-phosphate dehydrogenase deficiency. *Pediatrics* 70:364-369 (1982).
37. Valaes T, Doxiadis SA, Fessas P. Acute hemolysis due to naphthalene inhalation. *J Pediatr* 63:904-915 (1963).
38. Sidhu KS, Nash DF, McBride DE. Need to revise the national drinking water regulation for copper. *Regul Toxicol Pharmacol* 22:95-100 (1995).
39. Paustenbach DJ, ed. *The Risk Assessment of Environmental and Human Health Hazards: A Textbook of Case Studies*. New York:John Wiley and Sons, 1989.
40. Quinby GE, Clappison GB. Parathion poisoning. *Arch Environ Health* 3:438-442 (1961).
41. Fallon AE, Rozin P, Pliner P. The child's conception of food: the development of food rejections with special reference to disgust and contamination sensitivity. *Child Dev* 55:566-575 (1984).
42. Krause CM, Saarnio DA. Deciding what is safe to eat: young children's understanding of appearance, reality, and edibility. *J Appl Dev Psychol* 14:231-244 (1993).
43. Rozin P. Development in food domain. *Dev Psychol* 26:555-562 (1990).
44. Rozin P, Fallon A, Augustoni-Zisking M. The child's conception of food contamination sensitivity to "disgusting" substances. *Dev Psychol* 21:1075-1079 (1985).
45. Bicknell DJ. *Pica: A Childhood Symptom*. London:Butterworth and Co, 1975.
46. Danford DE, Huber AM. Pica among mentally retarded adults. *Am J Ment Defic* 87:141-146 (1982).
47. McAlpine C, Singh NN. Pica in institutionalized mentally retarded persons. *J Ment Defic* 30:171-178 (1986).
48. Stanek EJ III, Calabrese EJ, Mundt K, Pekow P, Yeatts KB. Prevalence of soil mouthing/ingestion among healthy children 1-6. *J Soil Contam* (in press).

Navy Environmental Health Center

NAVY ANNOUNCES OCCUPATIONAL HEALTH AND PREVENTIVE MEDICINE WORKSHOP

The Navy Environmental Health Center will host the Thirty-Ninth Navy Occupational Health and Preventive Medicine Workshop from March 28 to April 3, 1998, at the Town and Country Resort and Convention Center in San Diego, California.

The workshop's theme, "Knowledge, the Most Powerful Form of Prevention," will focus on wellness and prevention in the areas of occupational health and safety, preventive medicine, health promotion, environmental protection, and industrial hygiene. The Seventh Annual Health Promotion and the Fifth Annual Independent Duty Hospital Corpsman conferences will run concurrently.

Registration materials, hotel information, and the workshop advance program are available electronically on NEHC's homepage: www-nehc.med.navy.mil or call (757) 363-5508/5512. There is no registration fee for the conference.

Contact

Karen Murphy

2510 Walmer Ave. • Norfolk, VA 23510 • Phone: (757) 363-5451 • Fax: (757) 444-3672

E-mail: murphyk@nehc.med.navy.mil